

and has undoubtedly altered as time has passed, these figures nevertheless give some idea of the overall risk of serious disease in a population of some 50 million workers in an industrialized country. They therefore put such diseases into perspective. Figure 13-2 illustrates the certifications for asbestosis since 1964. It can be seen that the level has remained fairly static, around 120 to 150 per annum. The same figure also illustrates the number of deaths occurring in Britain in which asbestosis was mentioned on the medical certificate; there is a suggestion that a peak of around 200 per year may have been reached. Finally, the figures for death certificates mentioning mesothelioma reveal a steep climb from around 150 in 1968 to over 400 by 1978.⁵³ It is to be hoped that these figures will show a slower rate of climb within the next decade, reflecting the much tighter control over the importation and use of crocidolite in Britain since 1970.

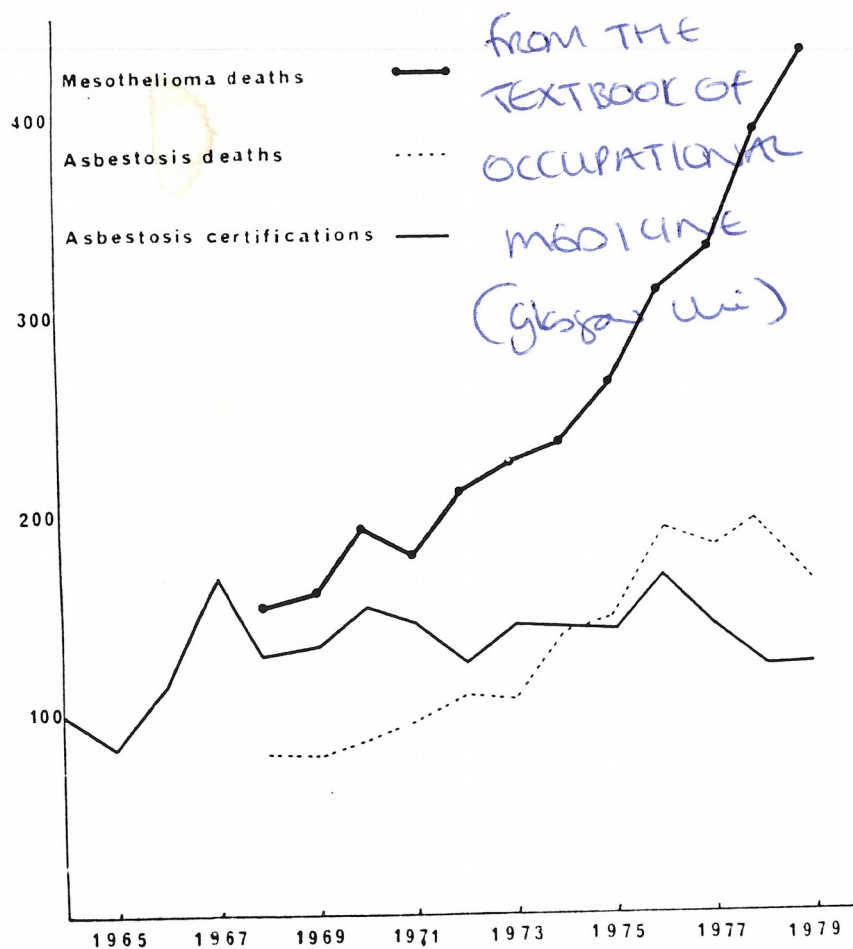


Figure 13-2. United Kingdom figures for certifications of asbestosis and deaths from asbestosis and mesothelioma, 1964-79.

initial recognition of the disease, the average age of death from asbestosis in Britain was about 30 and this had risen to 41 in the 1930s and 57 by 1965;^{51,55} it is currently in the mid 60s. The prevalence of asbestosis after 20 years of exposure in one factory in Britain fell from 80 to 3 per cent between 1929 and 1957.⁵⁵

It seems reasonable to suppose that through control of dust levels in the industry and medical surveillance of the work force at risk, together with reduced use of asbestos and increasing employment of substitute materials, asbestosis and asbestos-related lung cancer will become increasingly uncommon in the developed world. No such predictions can be made in the case of mesothelioma, except in countries where the use of crocidolite and amosite is to be abandoned.

ASBESTOSIS

MIS DIAGNOSIS

Clinical Features

The initial symptom of asbestosis is shortness of breath on exertion, often accompanied by a dry cough. This symptom develops only after several years of progressive pulmonary fibrosis. The dyspnea progresses even when the patient is removed from contact with asbestos, usually resulting in the development of cor pulmonale and death within 15 years of the onset of the disease. In the later stages, cough, sputum, and loss of weight are frequently present and the patient is often subject to recurrent respiratory infections. Before the advent of chemotherapy, tuberculosis and bronchopneumonia were responsible for a high proportion of deaths among patients with asbestosis, but this is no longer the case. The complications of bronchogenic, gastrointestinal, and pleural neoplasms are frequent causes of death, the patient's pulmonary fibrosis making these tumors even less likely to be resectable than in otherwise well patients.

Though in many instances asbestosis will progress even after cessation of exposure, there is some evidence that very early detection may prevent progression to a serious stage of fibrosis.^{56,57} The early detection of the disease is therefore a matter of great importance. Among workers exposed regularly to asbestos, the disease might be expected to develop within 15 to 20 years, though this clearly depends on dust levels, cases having been recorded as progressing from first exposure to death within 10 years. In some occupations, such as spraying, an exposure of as little as three years may result in the development of asbestosis. The cornerstones of early diagnosis are clinical examination, chest radiography, and lung function testing, and all three have at times been claimed to provide the most consistent chance of early, presymptomatic diagnosis.

The first physical sign of asbestosis is the presence of repetitive end-inspiratory crackles in the dependent parts of the lungs.^{58,59} These aus-

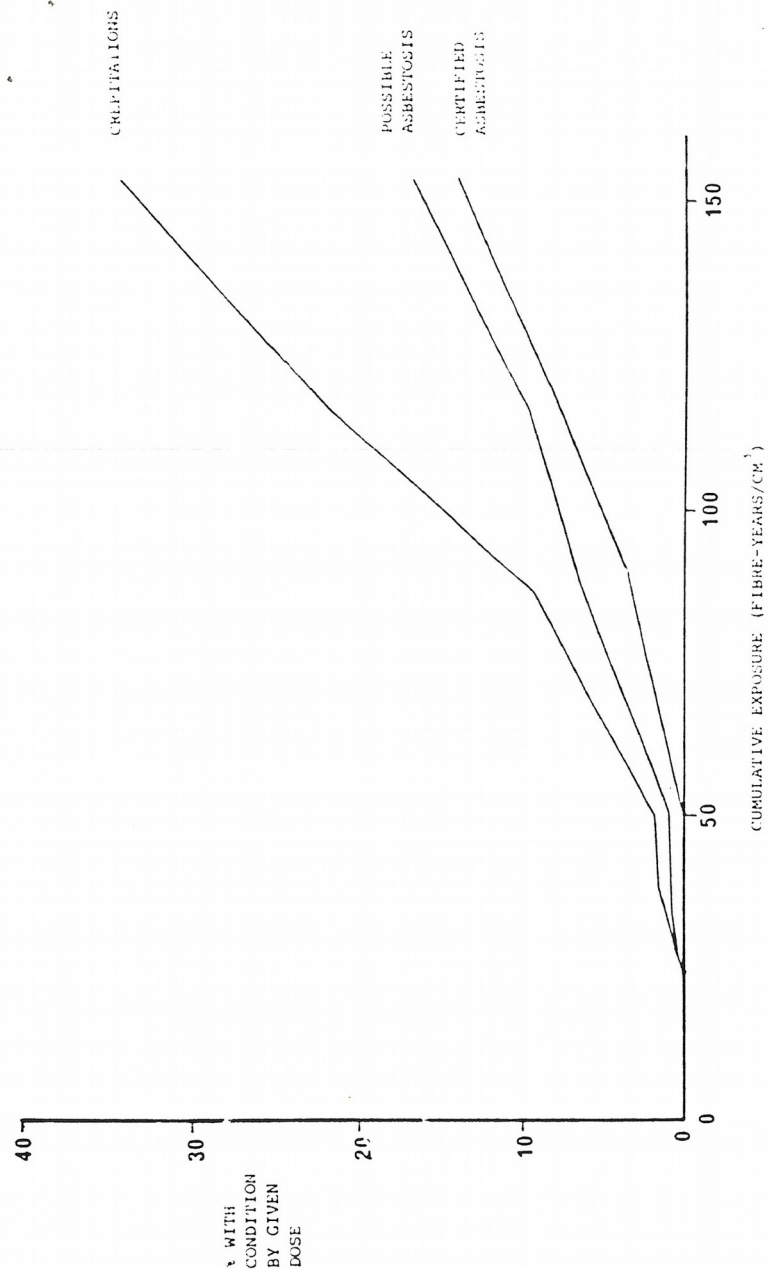


Figure 13-1. Observed risks of various indices of asbestosis among 379 men employed at least 10 years in an asbestos textile factory in relation to estimates of cumulative exposure (From Berry, G., et al., *Asbestosis: a study of dose-response relationships in an asbestos textile factory*, Br. J. Ind. Med. 36, 98, 1979).

not using asbestos directly may nevertheless be exposed to relatively high doses, for example, other workers close to the site of asbestos spraying or stripping, housewives washing overalls, and people living next to asbestos mills and factories in which hygiene is unsatisfactory. It is reasonable to assume that such people run risks similar to those of workers. In the absence of such exposure, it seems unlikely that a measurable increase in risk of asbestosis, lung cancer, or mesothelioma is likely to occur. However, this statement needs qualification, since exposure to asbestos may occur but not be recognized. For example, it seems likely that large numbers of people are at risk of mesothelioma in parts of the Cape Province in South Africa because of environmental contamination by crocidolite mine waste.¹⁴ Furthermore, it has recently been recognized that mesotheliomas may occur in certain parts of the world where fibrous minerals are found naturally in the rock and may be used for building and whitewash. Two such endemics have been described in separate parts of Turkey. In one, mesotheliomas and pleural plaques seemed to occur in relation to exposure to erionite, a very fine naturally occurring fibrous mineral^{15, 16} (see Chap. 12, p. 314), while in the other, the diseases were related to the use of whitewash and stucco containing tremolite.¹⁷ Such episodes point to the need for care and vigilance in the use and disposal of any material containing respirable fibers. Nevertheless, there seems little cause for anxiety in situations in which airborne asbestos levels are known to be low, as in public buildings,¹⁸ or in which minerals containing fibers are dumped or erode from pipes into drinking water. In these latter cases, an increased risk of gastrointestinal cancer among the users has been feared. Specific studies, however, showed no excess risk where asbestos cement pipes have been used for delivering the water supply^{19, 20} nor after cummingtonite-grunerite, a fibrous amphibole forming parts of the tailings from iron ore mining, was dumped in large quantities in Lake Superior.^{21, 22}

How Common is Asbestos-Related Lung Disease?

In most countries it is impossible to provide anything other than a crude guess as to the prevalence or incidence of asbestos-related disease. Estimates have varied from the overconfident to the overcautious, depending on the bias of the writer. In Britain an independent, government-run insurance system covers all employed people and provides, among other things, disability and death benefits for people suffering from recognized occupational diseases. Asbestosis, defined by strict criteria including the presence of some disablement, and mesothelioma have been included in this list of occupational diseases since 1931 and 1966, respectively. While clearly many factors determine whether a worker or his widow seeks and obtains such benefit, and also bearing in mind that the total number of workers and ex-workers at risk of the disease is not known

cultatory findings are probably related to abnormal lung deflation⁶⁰⁻⁶² and are indistinguishable from those heard in other forms of pulmonary fibrosis. It is possible, though not proved, that the height of lung over which the crackles may be heard is an indication of the severity of the fibrosis. As the disease progresses, tachypnea, cyanosis, and frequently clubbing of the digits may be found.^{63,64} Movements of the chest wall may become restricted and latterly signs of cor pulmonale, with tall jugular "a" waves, a right ventricular heave, and epigastric third and fourth heart sounds may be detected.

Radiographic Features

The inhalation of asbestos is associated with changes both in the lung and in the pleura. There has been difficulty in the classification of the lung changes because they are predominantly linear and irregular, as opposed to the characteristically nodular changes found in silicosis and coal workers' pneumoconiosis. However, as a result of the suggestions of Bohlig⁶⁵ and Sluis-Cremer and Theron,⁶⁶ international agreement has been reached on an adaptation of the ILO classification to include the irregular and pleural lesions of asbestos exposure in a general classification of radiological changes in pneumoconiosis. The resulting UICC/Cincinnati classification and the latest version of the ILO classification (1980)^{67,68} are described in Chapter 5, and the symbols of this classification are used in the following description.

As in any diffuse lung disease, the earliest radiographic signs of asbestosis are undefinable and little interobserver agreement may be obtained on their presence or absence. Such fine, irregular, linear shadows are classified as 0/1 or 1/0 for profusion and as type s. Larger irregular and linear opacities are classified as t and so-called coarse blotchy lesions as u. Whether there is any pathological significance attached to the differentiation of these opacities is not known, but this classification does provide a basis for further epidemiological and physiological studies, and one such prospective study has validated them as predictors of mortality.⁶⁹

Increasing profusion of the irregular shadows is indicated by an increased number up the scale from 1/0, 1/1, 1/2, 2/1, and so on, up to 3/- . At the stages between 2/2 and 3/3 the normal pulmonary vascular markings become obscured by the opacities. This classification avoids the use of terms implying pathological processes, such as fibrosis or honeycombing, and simply grades the film according to the size and profusion of the irregular opacities present. Standard films of the irregular opacities are available, and these are clearly necessary in order to achieve any form of international reproducibility.

Early evidence of asbestosis consists of linear shadows of varying thickness between less than 1 mm and 3 mm, most marked in the lower zones (Fig. 13-3). Increasing profusion of these markings results in a gradual obscuring of the vascular pattern and of the cardiac and diaphrag-



Figure 13-3. A. Chest radiograph of dock worker engaged for 20 years in repair work in ships' engine rooms, showing early changes of basal asbestosis and loss of volume, shown by depressed horizontal fissure, in right lower lobe. (Courtesy of Dr. J. Lyons.) B. Close-up view of left base of A, showing fine linear opacities of type S.

matic borders (Fig. 13-4). To a lesser extent nodular shadows may be present, and these may occasionally be the predominant opacity at an early stage. Ultimately there is a reduction in radiographic lung volume and the formation of cysts or honeycombing, combined with gradual increase in the cardiac size and dilatation of the proximal pulmonary arteries as cor pulmonale supervenes (Fig. 13-5). Throughout the course of the disease, with relatively few exceptions, the opacities are more profuse in the lower lung zones.

Only very occasionally do large masses, similar to those in complicated coal workers' pneumoconiosis, occur. In asbestosis, however, they have no particular predilection for the upper zones, being found in all parts of the lung.⁷⁰ The rare cases described in relation to rheumatoid disease have been basal in distribution.^{71,72} In the case of one of these patients, a follow-up report after his death 18 years later cast doubt on the role of asbestos in what appeared to have been widespread autoimmune disease.⁷³

Additional radiographic evidence of asbestos exposure, though not necessarily of asbestosis, may be obtained from the presence of pleural thickening⁷⁴ and pleural calcification⁷⁵ (Fig. 13-6). Most characteristically, bilateral obliteration of costophrenic angles and calcification on the diaphragm and in plaques overlying the posterior ribs may be found in asbestos-exposed subjects. In some investigations, conducted in areas where there is a known source of asbestos exposure, pleural plaques demonstrated at autopsy have been invariably associated with asbestos bodies in the lungs.⁷⁵ However, other investigations in areas where there is less of an asbestos hazard have failed to demonstrate a greater frequency of radiographic pleural lesions in asbestos-exposed subjects than in the control population.⁷⁶ Clearly there are other causes of pleural thickening and calcification, such as previous empyema, hemothorax, or pleurisy. If these conditions have been excluded, asbestos exposure is the most likely cause of pleural abnormalities and this is particularly so if the changes are bilateral.⁷⁷

As implied previously, the radiological appearances of asbestosis usually progress after exposure ceases, and in some cases may appear for the first time at this stage.⁷⁸ The rate of progression presumably depends on a combination of individual susceptibility and retained dose of dust in the lungs.⁷⁹

Pulmonary Function

The characteristic functional abnormalities in asbestosis are progressive reduction in vital capacity and total lung capacity, a normal or sometimes slightly raised residual volume, and reduction in diffusing capacity for carbon monoxide. As the disease advances, an increase in minute ventilation and arterial hypoxemia occur. Increased static recoil

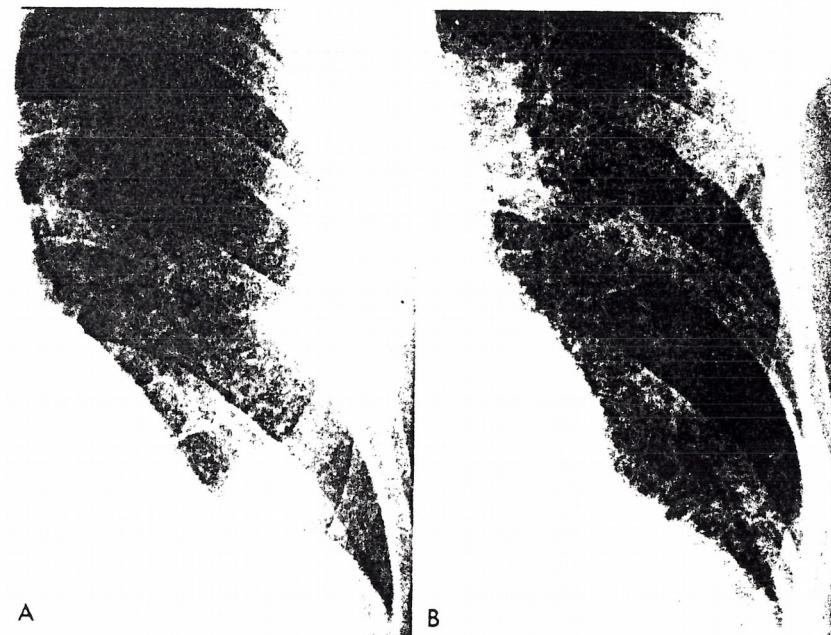


Figure 13-4. Development of asbestotic fibrosis in a shipyard demolition worker. A. Radiograph taken in 1967, showing only minimal linear and nodular changes at the bases. B. Radiograph taken in 1973, showing extensive basal fibrosis and honeycomb change. (Courtesy of Dr. J. Lyons.)

differ from those found in other industrial and nonindustrial forms of pulmonary fibrosis.^{38, 39}

More important than the functional definition of advanced disease is its detection at an early stage. Diffusing capacity seems to relate most closely to the degree of disability from asbestosis,⁴⁰ and attempts to detect the disease at a presymptomatic stage have concentrated on measurement of this and of vital capacity. Studies of lung function in relation to radiographic category^{41, 42} have suggested that decrease in vital capacity and increase in exercise ventilation are the most sensitive tests. Those studies compared the steady state diffusing capacity with the vital capacity and found the former less sensitive, but since it is affected by hyperventilation, the steady state test may not be as useful as the single breath technique. In contrast, other workers have demonstrated a fall in diffusing capacity at an early stage of the disease.^{37, 38} Fall in lung compliance parallels that in vital capacity.⁴²

Detailed studies of lung mechanics⁴³ in asbestos miners with normal chest radiographs have shown a reduction in vital capacity and an increase in static lung recoil pressures in those subjects with the heaviest exposure to asbestos. These subjects still had normal single breath and steady state diffusing capacities. Moreover, the more heavily exposed miners in general had lower maximal expiratory flow rates at a given transpulmonary pressure than the less heavily exposed group, indicating an increased upstream airways resistance requiring a higher pressure to achieve an equal flow. This work is in line with pathological studies that show that the early lesions in asbestosis are around the respiratory bronchioles, where they might be expected to cause obstructive disease of the small airways of the lung.⁴⁴ Indeed, a study of 23 workers exposed to high levels of chrysotile over a period of only five months has documented the development of airways obstruction rather than restriction as the dominant initial response in both smokers and nonsmokers.⁴⁵

The subject exposed to asbestos may therefore be expected to show a slight reduction in vital capacity and diffusing capacity and an increase in the static transpulmonary pressures at a relatively early stage in the disease process. Subsequent clinical deterioration will then be accompanied by further changes in these measurements, increase in resting and exercise ventilation, and decrease in total lung capacity. Arterial hypoxemia, initially on exercise and subsequently at rest, will appear, leading to the development of the complete picture of progressive alveolocapillary block and eventually cor pulmonale.

Pathology

The important pathological features of asbestos exposure are pulmonary fibrosis, the presence of asbestos bodies and fibers, and pleural plaques. In addition, massive fibrosis and chronic pleural thickening may be present, as may the complicating diseases, bronchial carcinoma and mesothelioma.

Asbestos Fibrosis

Macroscopically the appearance is of gray-white fibrosis, most extensive in the lower zones of the lungs (Fig. 13-7). Retraction of uninvolved bronchi and bronchioles may cause an appearance of honeycombing and sometimes bronchiectasis. Occasionally areas of massive fibrosis occur, and sometimes these cause retraction of and bullous formation in surrounding lung. This may be present in any part of the lung. The rounded nodules of Caplan's syndrome have occasionally been described^{72, 76} and seem to occur in the lower zones. The distribution of the fibrosis predominantly in the lower parts of the lungs in asbestosis helps in the differential diagnosis,⁴⁷ most other forms of pulmonary fibrosis being either more generalized, as in fibrosing alveolitis, or predominantly in the upper zones, as in sarcoidosis, tuberculosis, and allergic alveolitis. Further help in the differentiation may come from associated pleural thickening and calcification. Occasionally, however, cases of mixed asbestosis and silicosis may be found, as in some South African miners, where the fibrosis may be misleadingly distributed mainly in the upper zones.

Microscopically, the earliest lesion in both human and experimental asbestosis appears to be an alveolitis in relation to asbestos fibers deposited in alveoli, principally around respiratory bronchioles (Fig. 13-8). These alveoli fill with large cells which electron microscopical studies have shown to be the phagocytic lung macrophages.⁸⁸ These cells, some of which disintegrate, become organized, at first by deposition of fibrin and subsequently by collagen formation. Initially the more peripheral air spaces are uninvolved but eventually the fibrosis spreads out into distal air sacs and alveoli. Eventually intrabronchiolar spread results in a widespread network of fibrosis through the lungs, more marked at the bases, with dilatation of the uninvolved small airways.⁴⁹ Histologically, massive fibrosis differs from the more usual type only in being more confluent and extensive.

Asbestos Fibers and the Asbestos Body

The feature that most obviously differentiates asbestosis from other forms of lung fibrosis is the presence of asbestos bodies. These structures are yellow-brown in color and measure about 20 to 150 microns in length and 2 to 5 microns in width (Fig. 13-9). They have clubbed ends and a segmented or beaded appearance.⁹⁰ They appear to develop, at least initially, within lung macrophages, where there is an aggregation of small, 60Å granules around the phagocytosed part of the asbestos fiber. These granules probably consist of ferritin.⁸⁸ In addition, under the electron microscope there appears to be deposition of a lighter and nongranular material on the asbestos fiber. The coating of ferritin forms clumps and may eventually produce a body up to 5 microns thick.⁹¹

Such asbestos bodies may be found in the sputum of people exposed to asbestos, though they do not necessarily indicate asbestosis. They may also be demonstrated to be present in a high proportion of normal lungs at routine necropsy,⁹² by the use of ashed sections or special concentrating techniques, though routine hematoxylin and eosin staining reveals far

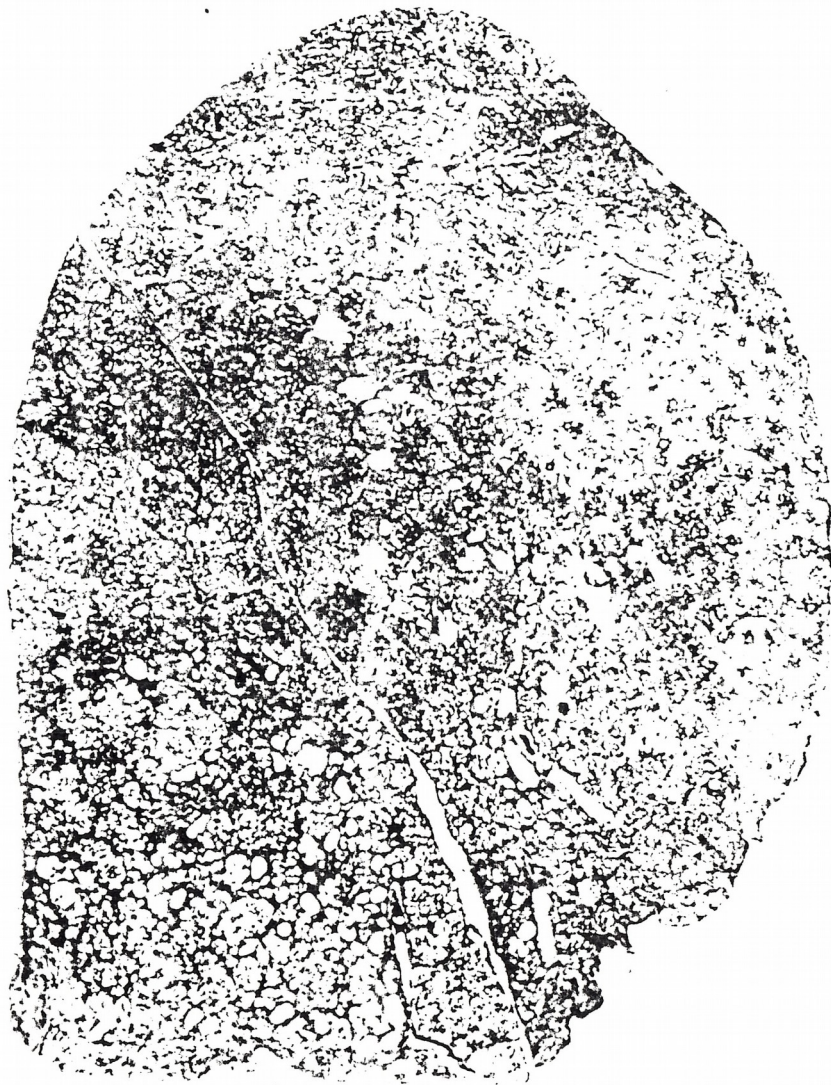


Figure 13-7. Whole-lung (Gough) section from patient with asbestosis, showing fibrotic changes and cyst formation in the lower lobe, with less marked changes in the lingula.



Figure 13-8. A. Low-power photomicrograph of lung of patient with severe asbestosis, showing masses of asbestos bodies and fibers situated in air spaces distorted by fibrosis. B. Medium-power photomicrograph of lung from an early case of asbestosis, showing relatively few asbestos bodies in a focus of interstitial fibrosis.



Figure 13-9. A. Two asbestos fibers, approximately 100 microns long, coated by iron-protein complex, from sputum of patient exposed to asbestos dust. B. Electron micrograph ($\times 5000$) showing asbestos bodies and fibers in lung tissue. Note the relative thinness of the fibers as compared to the bodies. Many fibers are seen to be uncoated, and their diameters would put them beyond the power of resolution of light microscopy.

fewer. There has been some dispute as to whether asbestos fibers invariably form the core of these bodies, as many other potentially respirable vegetable and mineral fibers may be found in the atmosphere.⁹³ However, electron microscopical studies with the use of microprobe analysis has confirmed that most such typical bodies do indeed contain asbestos, usually of the amphibole types, even in lungs obtained from the general population. Nevertheless, atypical or pseudo-asbestos bodies may form round a variety of other fibrous particles. It appears that in nonoccupationally exposed men, amosite and crocidolite most commonly form the cores of these bodies, while in women, tremolite, most likely from cosmetic talc, is found more frequently.⁹⁴⁻⁹⁶ Asbestos bodies may also be found in other parts of the body besides the lungs, forming round fibers transported by lung lymphatics into the circulation.⁹⁷ Studies of the frequency with which asbestos bodies may be found in the general necropsy population have shown no increase over 30 years in New York,⁹⁸ though there is evidence of an increase in Britain in relation to a cumulative increase in asbestos usage.⁹⁹ These differences may be related to differences in the amount of bonded, as opposed to potentially free, asbestos constituting the increase in usage.

Histological studies in patients with asbestosis show asbestos bodies aggregated together in areas of fibrosis (Fig. 13-8), and elastic staining can demonstrate that these were originally intra-alveolar.⁹⁹ Moreover, in areas of fibrosis, naked asbestos needles are also found, and are most easily demonstrated by polarized light. These average around 0.5 micron in diameter and 15 microns in length. Electron microscopy has shown them to be much more numerous than was originally suspected on the basis of light microscopy, and they can be shown in regions of fibrosis where asbestos bodies are not demonstrable (Fig. 13-9).¹⁰⁰ It is likely that the coating of fibers to form asbestos bodies renders them less liable to provoke fibrosis.¹⁰¹ Experimental studies on guinea pigs show that inhaled asbestos dust causes fibrosis, whereas asbestos bodies do not.¹⁰²

The use of an electron microscope reveals large numbers of asbestos fibers, even in the lungs of apparently nonexposed individuals. In one series from 21 urban dwellers, an average of 130 thousand fibers per gram of wet lung was found.¹⁰⁰ Most of these fibers are too small to form bodies or to be seen by light microscopy. Studies using light microscopy have shown that counts below 20,000 to 30,000 per gram dried lung represent the background level in people with no recorded exposure to asbestos, while counts above 100,000 can usually be related to a history of exposure.¹⁰⁴ Patients with mesothelioma usually have fiber counts of greater than 100,000 per gram dried lung, while in those with asbestosis the counts are usually over 3 million per gram.¹⁰⁵

Management

In view of the irreversible and often progressive nature of asbestosis, it is clear that the objective of industry and legislators should be to prevent

the disease. There is no evidence that asbestosis is curable, though early detection may be consistent with a relatively benign course. Industry should therefore institute regular screening of the work force, aimed at removing workers with early signs of disease from further exposure. Once the disease has developed, treatment can only be supportive, using symptomatic oxygen therapy and a cardiac failure regimen in the late stages. Smoking should be discouraged in view of the very high risks of lung cancer in smoking asbestotics. Where it is available, the patient should be encouraged to claim industrial injuries benefit as partial compensation for his disability. If the disease can be shown to be a consequence of the negligence of an employer (and this is by no means always possible), the patient or surviving relative may be in a position to claim compensation under the law of tort (see Chap. 1).

CARCINOMA AND ASBESTOS

As stated previously, workers exposed to asbestos have an increased risk of developing bronchial carcinoma. This risk appears to be related to the exposure dose as well as to the type of asbestos, being greater among workers exposed to crocidolite and amosite than among those exposed to chrysotile or anthophyllite. The evidence suggests that there is an interaction between asbestos exposure and cigarette smoking. Selikoff and co-workers found no cases of bronchial carcinoma in nonsmokers, whereas the risk increased with increasing categories of exposure and cigarette smoking, such that a heavily exposed smoker had a risk 9 times that of a nonexposed smoker.¹³ It has been calculated that the effects of the two carcinogens act in a multiplicative manner.¹⁴

Many studies have also shown an increased risk of other neoplasms in asbestos workers, though the risks differ in different studies.¹⁰⁷ There does seem to be an excess of intestinal carcinoma in some circumstances,¹⁰⁸ but the association of asbestos exposure with laryngeal carcinoma is dubious.¹⁰⁹

Compared with bronchial carcinoma in the general population, the tumors occurring in an asbestos-exposed group seem to include an excess of the adenocarcinoma cell type. As both asbestosis and lung cancer are related to the exposure dose, the two occur together frequently. The percentage of patients with asbestosis who die of carcinoma has in fact risen from 17 per cent at the time of Merewether's report⁹ to over 50 per cent.¹¹⁰ However, it is possible that the carcinoma could develop before clinical evidence of asbestosis became apparent. Such cases may give rise to problems in deciding whether compensation is due, in life, to the subject and, after death, to his relatives. The presence of a history of exposure and radiographic features of asbestosis would be required in life, and, after death, the demonstration with relative ease of asbestos bodies or fibers and also asbestotic fibrosis. Clearly there is an ill-defined

borderland here, each case having to be decided on its merits (Fig. 13-10 and 13-11). Management of the carcinoma should be along standard lines. The presence of asbestosis will reduce the likelihood of resectability, but surgery probably remains the treatment of choice for all resectable tumors. Chemotherapy may eventually establish a place for itself in the management of oat-cell tumors, though to date it has only shown itself capable of prolonging life at a price of not inconsiderable side effects. In many cases, especially with oat-cell and epidermoid tumors, radiotherapy is the treatment required for symptomatic relief.

PROVES CHEMOTHERAPY IS
MESOTHELIOMA

In 1960, Wagner and his colleagues¹¹ from the North-West Cape of South Africa reported 33 cases of the rare pleural or peritoneal tumor, mesothelioma. The geographical distribution suggested an environmental factor, in particular, exposure to North-West Cape crocidolite asbestos. Cases were not found in the chrysotile or amosite mining areas of South Africa nor in the crocidolite area adjacent to the amosite deposits. Development of the tumor was not apparently related to the dose of asbestos inhaled, mesotheliomata being found in subjects with or without asbestosis, as well as in people who had been exposed in an environmental rather than an occupational setting. An interesting finding was the very long latent period, a mean of 10 years, between initial exposure and subsequent development of the tumor.

Shortly afterward, similar tumors were shown to occur in relation to asbestos exposure in Britain and the United States, as well as in the crocidolite-producing area of Australia. The relationship between mesothelioma and asbestos exposure has been investigated by two different epidemiological approaches. First, studies of populations of asbestos textile, shipyard, and insulation workers have shown a higher than expected number of cases, and second, surveys of patients with mesothelioma have shown that a high proportion give a history of asbestos exposure, albeit often for a short period, in the past. Elmes and Wade¹¹⁶ found 45 cases of mesothelioma in Belfast, a shipbuilding and repairing area, and noted that there was a significantly higher exposure to asbestos among patients than among controls. Moreover, the lungs of 75 per cent of the patients, as opposed to 25 per cent of the controls, contained asbestos bodies. Most of these patients had been exposed to asbestos intermittently rather than continuously. Selikoff and co-workers¹¹² described 22 cases of mesothelioma, 6 pleural and 16 peritoneal, among New York insulation workers, the figure representing about 6 per cent of all deaths. It was shown that these workers would have had little if any exposure to crocidolite, since chrysotile and amosite had been used almost exclusively in the insulation trade. More recent work, discussed previously, has shown reasonably convincingly that there is a gradient from crocidolite to amosite to

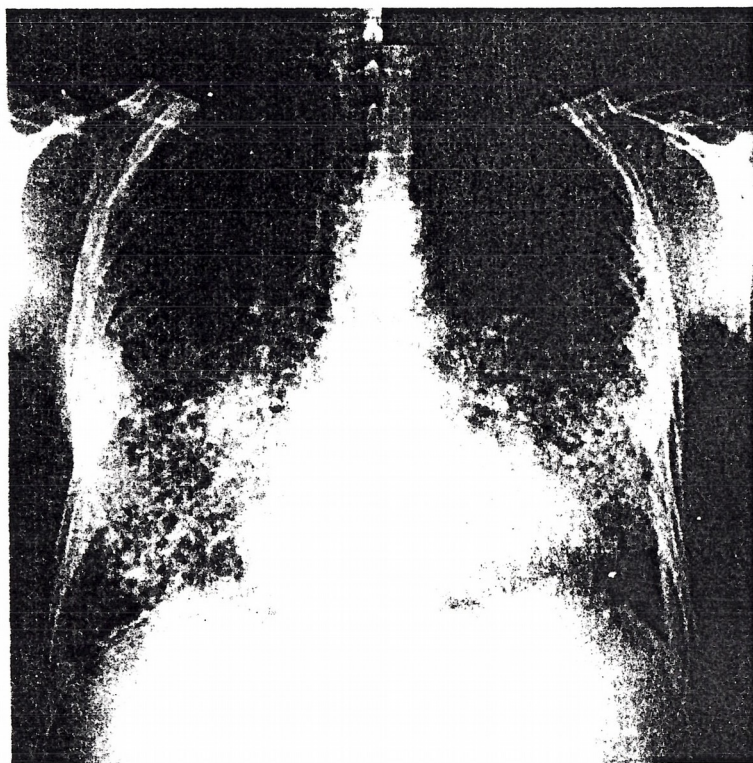
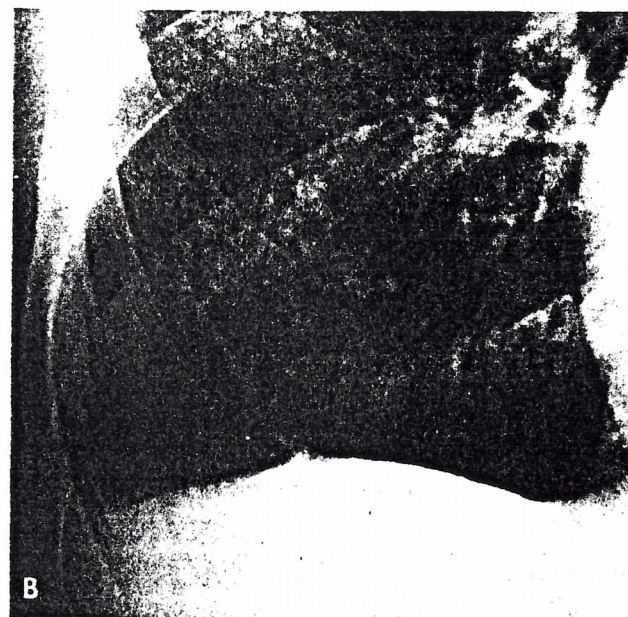


Figure 13-5. Radiograph of insulation worker who presented with breathlessness, finger clubbing, and evidence of cor pulmonale, showing advanced asbestos fibrosis, obliterated costophrenic angles, and calcification over right hemidiaphragm.



A



B

Figure 13-6. A, Large, noncalcified plaques (arrowed) in a shipyard worker exposed to asbestos. In addition, scant parenchymal asbestotic lesions are present. (Courtesy of Dr. J. Evans.) B, Close-up view of the right lower zone of chest film of another subject. A well-